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# Intensity and physiological strain of competitive ultra-endurance exercise in humans

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**Abstract**

The aim of this study was to determine the magnitude and pattern of intensity, and physiological strain, of competitive exercise performed across several days, as in adventure racing. Data were obtained from three teams of four athletes (7 males, 5 females; mean age 36 years, $s = 11$; cycling $\dot{V}O_{2\ peak}$ 53.9 $ml \cdot kg^{-1} \cdot min^{-1}$, $s = 6.3$) in an international race (2003 Southern Traverse; 96–116 h). Heart rates (HR) averaged 64% (95% confidence interval: $\pm$ 4%) of heart rate range [$\%HRR = (HR - HR_{min})/(HR_{max} - HR_{min}) \times 100$] during the first 12 h of racing, fell to 41% ($\pm$4%) by 24 h, and remained so thereafter. The level and pattern of heart rate were similar across teams, despite one leading and one trailing all other teams. Core temperature remained between 36.0 and 39.2°C despite widely varying thermal stress. Venous samples, obtained before, during, and after the race, revealed increased neutrophil, monocyte and lymphocyte concentrations ($P < 0.01$), and increased plasma volume (25 $\pm$ 10%; $P < 0.01$) with a stable sodium concentration. Standardized exercise tests, performed pre and post race, showed little change in the heart rate–work rate relationship ($P = 0.53$), but a higher perception of effort post race ($P < 0.01$). These results provide the first comprehensive report of physiological strain associated with adventure racing.

**Keywords:** *Physiological strain, exercise intensity, adventure racing, sleep deprivation*

## Introduction

Adventure racing imposes unique stress on competitors, as races can extend over 100 h, with top competitors typically reporting only 1 h of sleep in each 24 h. Teams of competitors navigate a novel course through stages of, for example, jogging/trekking, kayaking, mountain biking, roped ascent/descent, and coasteering, in the shortest possible time. Teams normally include at least one female competitor. Within-team pace differences due to fitness, injury, fatigue or technical differences are minimized through load sharing, drafting, and towing.

There is a lack of research examining very prolonged, multi-day, semi-continuous exercise due to problems of maintaining compliance and vigilance in laboratory-based protocols for extended periods (Krueger, 1991), and poor validity of the laboratory environment for prolonged competitive exercise. Military-based studies have reported effects of multi-day operations (36–120 h), including effects on physical performance (Myles, Eclache, & Beury, 1979; Myles & Romet, 1987; Nindl *et al.*, 2002), immune and hormonal responses (Bøyum *et al.*, 1996; Brenner, 2000; Gomez-Merino, Chennaoui, Burnat, Drogou, & Guezennec, 2003; Opstad & Aakvaag, 1983; Opstad, Oktedalen, Aakvaag, Fonnum, & Lund, 1985), and thermoregulation (Castellani *et al.*, 2003). However, these studies have not addressed the absolute volume of competitively constrained work in an adventure race, and often included more rest and/or sleep. Accordingly, the multi-day rate of energy expenditure of adventure racing (Doel, Hellemans, & Thomson, 2005) appears to be higher than that reported in military-based studies (Forbes-Ewan, Morrissey, Gregg, & Waters, 1989; Hoyt *et al.*, 1991), elite cycle tours, such as the Tour de France (Westerterp, Saris, van Es, & ten Hoor, 1986), or Polar expeditions

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(Stroud, Coward, & Sawyer, 1993; Stroud *et al.*, 1997). The lack of information on adventure racing is unsurprising due to its relatively recent development, the typically remote locations and competitive nature of events, the secrecy over the course until only hours before race start, and limitations associated with field-based testing. Development in measurement technology for in-field data collection is becoming more feasible and less intrusive. The literature on adventure racing is limited to reporting illness and injuries (Fordham, Garbutt, & Lopes, 2004; Melody *et al.*, 1999; Townes, Talbot, Wedmore, & Billingsly, 2004), cardiac (left ventricular) dysfunction after racing (Ashley *et al.*, 2006), and a preliminary report of selected physiological factors (Walmsley, Hopkins, Cotter, & Rehrer, 2002). The latter study was based on four athletes in the 2000 Southern Traverse adventure race, and the authors reported that peak heart rate fell from 140 beats $\cdot$ min$^{-1}$ on day 1 to 100 beats $\cdot$ min$^{-1}$ on day 5, and that core temperature and cognitive assessment results were relatively stable. Thus, the physiological strain and exercise pattern of multi-day, sustained, and competitive exertion in humans remains largely unknown. This study provides the first comprehensive description of the physiological strain of multi-day competitive exertion in an international adventure race.

## Methods

### *Participants*

Three teams of four athletes were studied before, during, and after the race. The teams varied in fitness and experience (Table I). The competitors of one team had competed internationally in adventure racing (Team A: mean cycling peak oxygen uptake [$\dot{V}O_{2peak}$] = 59.1 ml $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$, $s = 6.5$; one female member), a second team had extensive Southern Traverse experience (Team B: mean cycling $\dot{V}O_{2peak}$ = 51.8 ml $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$, $s = 5.8$; one female member), and a third team were adventure racing novices (Team C: mean cycling $\dot{V}O_{2peak}$ = 49.6 ml $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$, $s = 3.2$ for the three female members). The study was approved by the University of Otago Ethics Committee, and all participants provided their informed, written consent.

### *Course*

The 2003 Southern Traverse adventure race took place in Eastern Otago, New Zealand, near the University of Otago; the 2003 race was the twelfth. The Southern Traverse is the world's second oldest adventure race, and is part of the Adventure Racing World Series.

The full course (completed without navigational errors) was 411 km, divided into 14 stages (Figure 1). Competitors trekked/jogged (3, 27, 70, 14 km), kayaked (22, 35, 25, 28 km), mountain biked (50, 52, 40, 17 km), and coasteered (16, 12 km). Each stage was separated by a transitional area where competitors changed exercise mode and received food and attention from support crew. Teams failing to reach designated locations within a pre-disclosed time were placed on a shortened course, so that all finishing teams would do so within $\sim$24 h of one another. Scientific testing occurred at the transitional

Table I. Characteristics of the members of the three teams ($n = 12$) taking part in the research before, during, and after the 2003 Southern Traverse adventure race.

| | | | | | Cycling $\dot{V}O_{2peak}$ | | |
|---|---|---|---|---|---|---|---|
| ID # | Sex | Age | Mass (kg) | Height (m) | (litres $\cdot$ min$^{-1}$) | (ml $\cdot$ kg$^{-1}$ $\cdot$ min$^{-1}$) | Adventure racing experience |
| **Team A** | | | | | | | |
| 1 | M | 31 | 92.0 | 1.84 | 4.8 | 51.9 | International |
| 2 | M | 44 | 77.2 | 1.78 | 4.8 | 61.9 | |
| 3 | M | 38 | 72.1 | 1.69 | 4.8 | 66.7 | |
| 4 | F | 31 | 66.6 | 1.75 | 3.7 | 55.8 | |
| **Team B** | | | | | | | |
| 5 | M | 41 | 74.1 | 1.76 | 4.0 | 54.3 | Extensive Southern Traverse |
| 6 | M | 53 | 73.4 | 1.85 | 4.2 | 57.3 | |
| 7 | M | 53 | 96.0 | 1.79 | 5.0 | 52.0 | |
| 8 | F | 34 | 79.0 | 1.81 | 3.5 | 43.7 | |
| **Team C** | | | | | | | |
| 9 | F | 29 | 69.1 | 1.61 | 3.4 | 48.2 | Novice |
| 10 | F | 21 | 60.5 | 1.62 | 3.2 | 53.3 | |
| 11 | M | 26 | 94.7 | 1.91 | | | |
| 12 | F | 25 | 66.0 | 1.61 | 3.1 | 47.3 | |
| Mean | | 36 | 76.7 | 175 | 4.0 | 53.9 | |
| *s* | | 11 | 8.9 | 11 | 0.7 | 6.3 | |

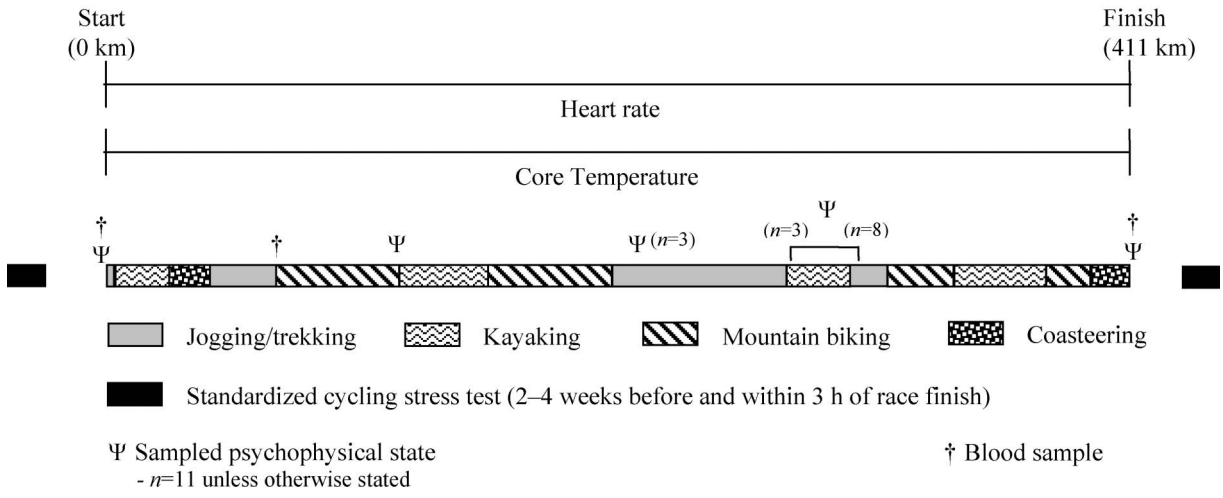


Figure 1. Schematic representation of the different stages of the 2003 Southern Traverse adventure race, and when physiological and psychophysical strain measurements were recorded before, during, and after the race.

areas and during selected stages of the race. Ambient temperature was obtained from two national meteorological service (NIWA Science, New Zealand) weather stations in the race area.

*Measurements and protocol*

Figure 1 outlines the race sequence and experimental protocol. Maximum heart rate ($HR_{max}$) and $\dot{V}O_{2peak}$ were determined 3–5 weeks beforehand on an electro-magnetically braked cycle ergometer (Rodby, Elektronik AB, Södertälje, Sweden), using a standard progressive exercise test: 30 W every minute, beginning at 100 W for females and 150 W for males, with respiratory gas analysis (Sensormedics, 2900Z BXB, Sensormedics Corp., Yorba Linda, CA, USA). Minimum heart rate ($HR_{min}$) was determined from the lowest heart rate measured either in the hours before the race or during the race (i.e. including sleep). Participants' relative exercise intensity (i.e. work rate) during the race was measured from heart rate (Åstrand & Rodahl, 1986), as the percentage of their heart rate range [$\%HRR = (HR - HR_{min})/(HR_{max} - HR_{min}) \times 100$]. Heart rate was measured at 1-min intervals throughout the race (Polar® S610i and S720i monitors, Polar Electro, Kempele, Finland).

Heart rate recorded during the race was validated against possible pre- to-post race shifts by measuring the heart rate–work rate relationship during a standardized exercise test. This test was performed once on a subsequent day to the $\dot{V}O_{2peak}$ test 2–4 weeks before, and within 3 h of completing, the race. Participants completed 8-min blocks of seated rest and cycling at 25% followed by 50% of the pre-race peak power output obtained in their $\dot{V}O_{2peak}$ test. These work rates corresponded to 34 and 58% of $\dot{V}O_{2peak}$ respectively. Heart rate was recorded using three-lead electrocardiography (MRL Satellite System II Monitor, Medical Research Laboratories Inc., Illinois, USA), and measured from the R–R interval of the QRS complex throughout the final 2 min of each 8-min period. Participants rated their perception of exertion on the Borg 10-point scale (Borg, 1982) during these 2 min of each 8-min block.

Core temperature was measured at 1-min intervals throughout the race using gastrointestinal thermometer pills (CorTemp pill sensors, Human Technology Industries Inc., Florida, USA), transmitting to portable loggers (BCTM5, Fitsense, USA). Teams A and B had two loggers each that were rotated around team members during the race. Once participants had excreted a pill, another sterile pill was ingested and the logger calibrated to the currently ingested pill. Factory calibration of pills was $\pm$ 0.1°C.

Ratings of perceived exertion and thermal discomfort were obtained periodically during the race (Figure 1) using a 5-point Likert scale (Likert, 1932), ranging from "nothing at all" (1) to "extreme" (5). A 5-point scale was used to maintain measurement consistency with a parallel assessment of mood (Brunel Mood Scale; Terry, Lane, & Fogarty, 2003). Participants were familiarized with the questionnaire before the race.

Venous blood ($n = 11$) was obtained 24 h before the start of the race (more than 6 h fasted), at a known transition area early in the race (15–30 h), and at the finish (96–116 h). Venous blood samples were drawn, without stasis, from a suitable forearm vein via a 20-gauge needle into a 10-ml syringe following 3–5 min of seated rest. Samples were transferred into EDTA-preservative tubes, and a whole-blood portion of the 10-ml sample ($\sim$2 ml)

was analysed by a commercial laboratory for white blood cell differential using an automated haematology analyser (Sysmex XE-2100, Sysmex Corporation, Japan). Changes in plasma volume were estimated from changes in haematocrit and haemoglobin concentrations (Dill & Costill, 1974). Arginine vasopressin concentration was measured using a radio-immuno assay/RIA, Endolab, Christchurch, New Zealand). Within-assay coefficients of variance were between 6 and 12% across the range of arginine vasopressin concentration observed. Pre- and post-race blood sodium concentrations were measured in 6 of the 11 participants from serum samples obtained after 6 min of sitting following the standardized exercise test (Hitachi 911 analyser with ISE technology, Roche, New Jersey, USA). Urine samples were obtained with the blood samples pre and post race. Urine specific gravity was measured using a portable refractometer (Atago, Astra Zeneca Pty Ltd., Japan).

The distributions of total body fluid, intracellular and extracellular volumes were estimated using multi-frequency bio-impedance analysis (4-frequency, 8-polar tactile-electrode; InBody 3.0, Biospace, Seoul, Korea). The bio-impedance estimates of total body fat, protein, and mineral masses were also recorded. Participants ($n = 11$) stood barefoot on the device, twice under fasted ($>6$ h) and rested ($>12$ h) conditions, 2 days before racing and on the morning following racing, and once within 60 min of finishing the race.

### *Data analyses*

The pattern of cardiovascular strain for a team was measured as the mean percent heart rate range (95% confidence interval) across the four members. Outliers in heart rate data were removed from the mean calculation by comparing individuals' heart rate to those of other team members. A recorded heart rate was defined as an outlier when it was drastically out of character with intrapersonal measurements before and after the ''abnormal'' heart rate recording (e.g. 240 beats $\cdot$ min$^{-1}$), and when the recording did not match the typical response of other team members. Data that did not meet both of these criteria for exclusion remained part of the mean calculation. The mean calculation for each team was not always from all four members because of field-testing limitations preventing acquisition of a complete set of heart rate data from each individual for every minute of the race. The pattern of thermoregulatory strain was determined from the recorded core temperature of participants. Individuals' data were plotted to show body core temperatures within each team (A and B) across the hours of racing. Data that changed more than 0.5°C between subsequent minutes were deemed erroneous and removed.

The magnitude of race-mediated shifts in the heart rate–work rate relationship and the perceived exertion–work rate relationship were analysed with a two-way repeated-measures analysis of variance (ANOVA; SPSS 13.0, SPSS Inc., Illinois, USA), using individuals' heart rate and perceived exertion recorded from the three standardized exercise periods (rest, 25% and 50% peak power output) before and after the race. Changes in white blood cell counts were analysed using a repeated-measures ANOVA with planned comparisons (Bonferroni corrections) applied against the baseline values. The likely range for the true value is shown by the 95% confidence interval. A Pearson correlation was performed to examine relationships between the change in plasma volume and the initial haemoglobin concentration and arginine vasopressin concentration. Statistical significance for each analysis was set at $P < 0.05$. Results are presented as means and standard deviations unless otherwise stated.

## Results

Teams A and B completed the full course (411 km) in 96 and 108 h, respectively; Team C completed a shortened course (341 km) in 116 h. The recorded ambient temperatures (minimum–maximum) were: 5.2–22.3°C on day 1; 11.5–22.2°C on day 2; 7.1–11.4°C on day 3; 4.4–13.0°C on day 4; and 2.6–16.8°C on day 5. Racers experienced snow and hail on days 3 and 4 while at altitudes of $\sim$1000 m. Ambient temperature was recorded from weather stations at altitudes of 0 and 280 m. One member of Team A retired 36 h into the race (acute illness); the remaining members completed the race. Participants slept for an average of 13 min ($s = 12$; range 0–30) during the first 24 h, and then 1 h 36 min ($s = 29$ min; range 0:57 to 2:01 h:min) in each subsequent 24-h period (estimated from daily self-report logs).

### *Exercise intensity*

*In race.* Heart rates averaged 64% (95% confidence interval [CI]: 60 to 68%) of heart rate range during the first 12 h of racing, fell to 41% (95% CI: 38 to 45%) by 24 h, and remained at approximately this level thereafter (Figure 2). The pattern and magnitude of cardiovascular strain during the first day were consistent across the three teams despite one team leading and one team trailing the other 29 teams. Across the 5 days, athletes spent $\sim$40% of their time exercising at moderate intensity (40–60% HRR; Figure 3). On day 1 they were otherwise mostly

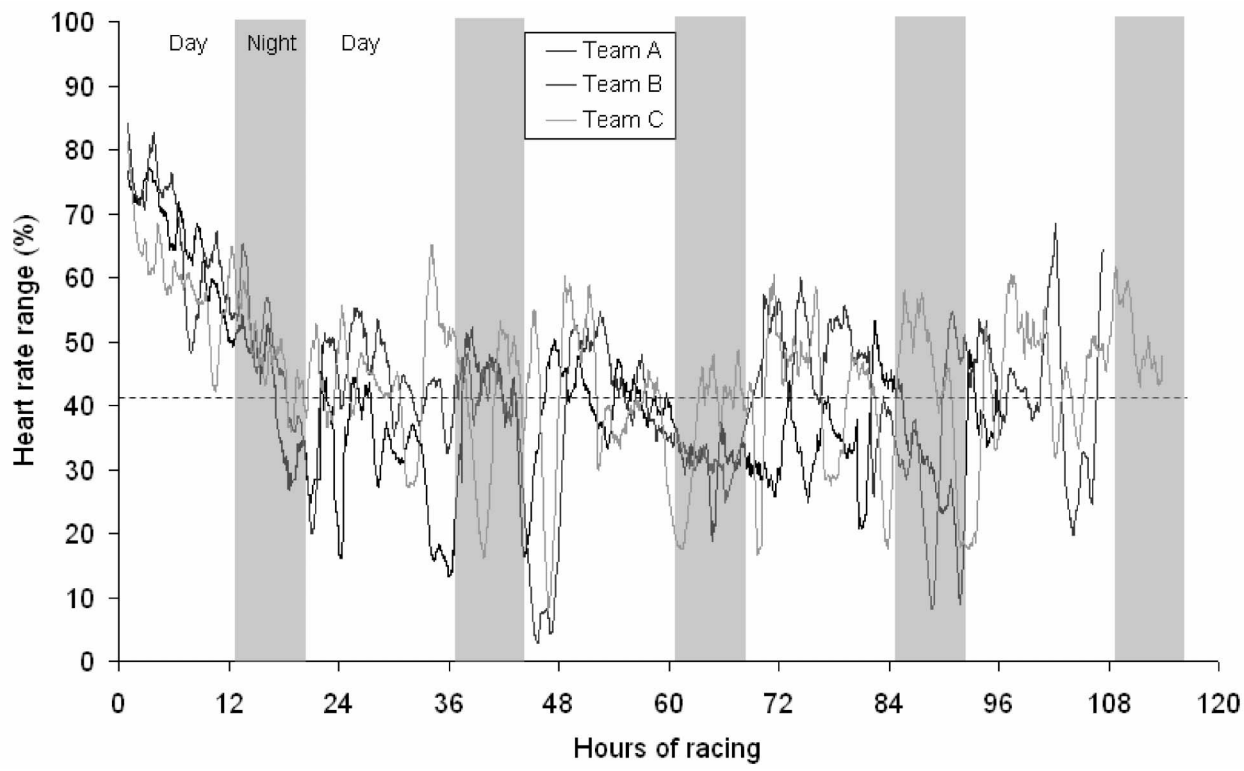


Figure 2. The pattern of cardiovascular strain for each team throughout the race, represented by the mean proportion of heart rate range (%HRR = (HR − $HR_{min}$)/($HR_{max}$ − $HR_{min}$) × 100). The means are across the four members of each team, with a 1-h rolling time average. Shaded areas identify night periods (21:30–05:00 h).

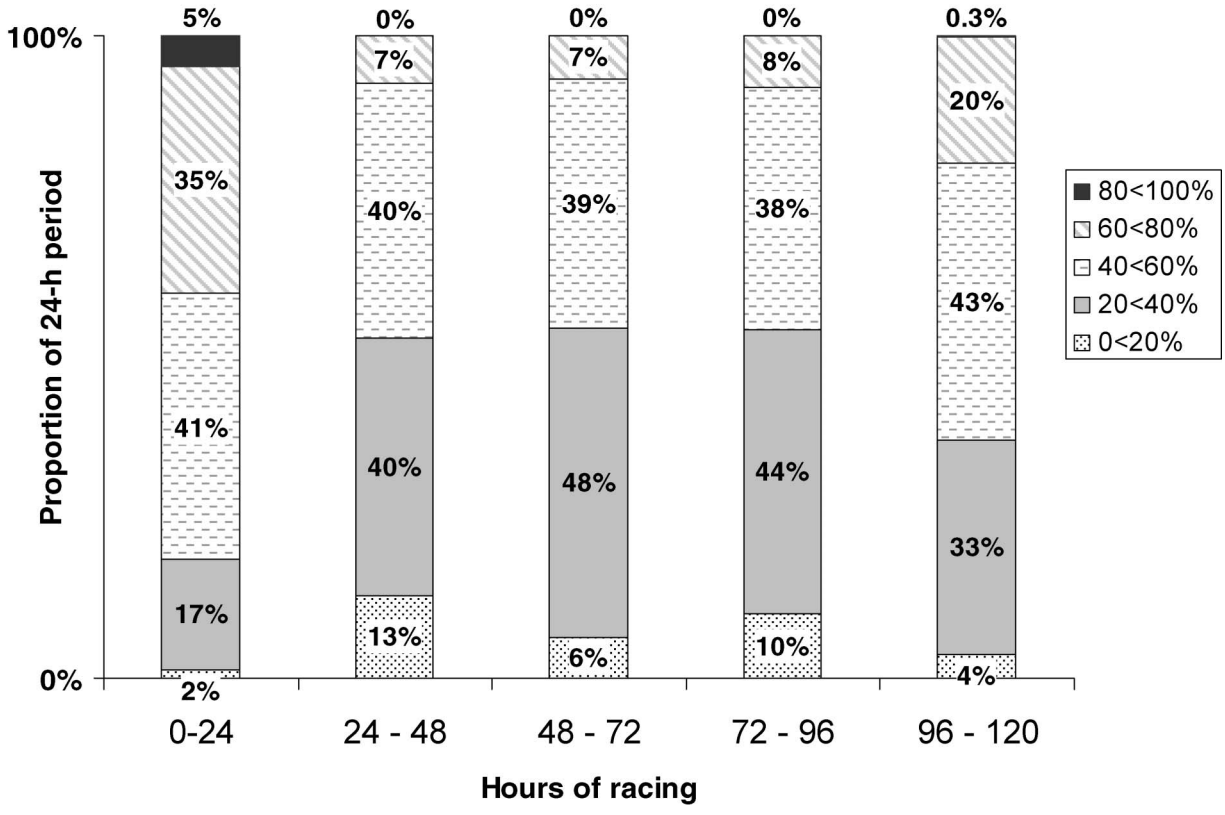


Figure 3. The percentage of time spent in relative heart rate intensity zones within each 24-h period. Data presented are the mean of the three teams.

above this intensity, and on days 2–5 were otherwise mostly below it.

*Standardized work tests (pre and post race)*. There was no difference in mean hearts pre and post race during the standardized workloads ($P = 0.53$): 68 beats · $min^{-1}$ ($s = 15$) and 68 beats · $min^{-1}$ ($s = 8$) at rest before and after the race respectively; 99 beats · $min^{-1}$ ($s = 8$) and 106 beats · $min^{-1}$ ($s = 20$) at 25% peak power output before and after the race respectively; and 138 beats · $min^{-1}$ ($s = 16$) and 139 beats · $min^{-1}$ ($s = 24$) at 50% peak power output before and after the race respectively. There was no interaction between the time of test (pre vs. post) and workload (rest, 25%, 50%; $P = 0.29$). Therefore, heart rate remained a valid indicator of work rate across these workloads.

Ratings of perceived exertion at 25% and 50% standardized workloads were greater after the race ($P < 0.01$), despite the stable heart rate response. The increase in perception was workload dependent ($P = 0.04$); exertion ratings increased by almost 1 scale point at 25% peak power output (from 2.7 ''weak–moderate'' to 3.5 ''moderate–somewhat strong'') and by almost 2 scale points at 50% peak power output (from 4.9 ''strong'' to 6.7 ''very strong''). Thus, in contrast to the stable heart rate–work rate relationship pre to post race, perceived exertion increased relative to work rate at post-race testing and was related to intensity.

*Core temperature*

Figure 4 shows that core temperature exceeded 39.0°C for only 1.5 h (<1%), remaining mostly at 38.0–39.0°C (22%) or 37.0–38.0°C (67%), and reached a minimum of 36.0°C. These findings occurred under conditions in which athletes were exposed to a variety of endogenous and exogenous thermal stresses (e.g. running in wetsuits, walking and cycling in snow).

*Perceived strain*

Ratings of perceived physical exertion (scale 1–5) on days 2–5 of the race varied from 3 ''moderate'' to 4 ''quite a bit''. Ratings of thermal discomfort (too cold or hot) ranged from 2 ''a little'' to 3 ''moderate'', although data were not obtained at thermally stressful times such as during a snow storm on the third night. Individual results varied between and within teams throughout the race.

*Fluid measures*

Plasma volume increased 10% ($s = 7$) by 15–30 h of racing ($P < 0.01$; Figure 5), despite a 2% decrease ($P < 0.01$) in mean cell volume. Correspondingly, the reticulocyte count had dropped 10% ($s = 15$) ($P = 0.01$) by this same time. At race finish, plasma volume had increased by 25% ($s = 10$) ($P < 0.01$) and the reticulocyte count had fallen by 22% ($s = 30$) ($P = 0.04$). This hypervolaemic response was independent of athletes' initial haemoglobin concentration ($r = 0.09$, $P = 0.79$), but was correlated with arginine vasopressin concentration ($r = 0.70$; $P < 0.01$). However, no arginine vasopressin concentration exceeded 3 pmol · $l^{-1}$ and end-race urine specific gravity was not high (1.018, $s = 0.006$). Blood serum sodium concentration measured at the end of the race in six athletes was 138.7 mmol · $l^{-1}$ ($s = 0.8$) compared with 138.7 mmol · $l^{-1}$ ($s = 2.3$) before the race, thus blood sodium concentration was maintained despite the large plasma volume expansion.

Intracellular fluid volume, as measured by bio-impedance under standardized conditions both pre and post race, was stable across the race (from 32.1 to 32.0 litres; $P = 0.11$), while extracellular fluid volume increased 7% ($s = 3$) (from 14.6 to 15.5 litres; $P < 0.01$; see Figure 6). Bio-impedance analysis showed a 1.6-kg ($s = 0.4$) ($P < 0.05$) decrease in fat mass, while body mass decreased by only 0.9 kg ($s = 0.2$) ($P < 0.05$).

*Immune measures*

Baseline plasma concentrations of white blood cells were within normal ranges (Figure 7). Early in racing (15–30 h), average plasma concentrations of neutrophils, lymphocytes, and monocytes increased ($P < 0.01$; Figure 7) by 166% (95% CI: 125 to 208%), 42% (95% CI: 21 to 63%), and 131% (95% CI: 84 to 178%), respectively. At race completion, neutrophils and monocytes remained elevated by 77% (95% CI: 54 to 101%) and 58% (95% CI: 30 to 85%), respectively ($P < 0.01$); lymphocytes on average were elevated, but not consistently so (up 8%; 95% CI: −6 to 30%; $P = 0.36$). These non-specific immune marker changes were more marked in individuals from Team A ($8.92 \times 10^9 \cdot l^{-1}$, $s = 0.67$ and $1.28 \times 10^9 \cdot l^{-1}$, $s = 0.18$, respectively) and Team B ($8.36 \times 10^9 \cdot l^{-1}$, $s = 1.40$ and $1.39 \times 10^9 \cdot l^{-1}$, $s = 0.21$, respectively) than Team C ($5.43 \times 10^9 \cdot l^{-1}$, $s = 1.42$ and $0.68 \times 10^9 \cdot l^{-1}$, $s = 0.20$, respectively). This was possibly due to differences in elapsed race time and teams' exercise intensities in the hours leading into the transition area where the blood samples were obtained. Plasma concentrations of basophils and eosinophils were lower than baseline early in racing, by 33% (95% CI: 51 to 15%; $P = 0.31$) and 57% (95% CI: 70 to 43%; $P < 0.01$) respectively, and remained low at race completion – basophils by 43% (95% CI: 65 to 20; $P = 0.06$) and eosinophils by 31% (95% CI: 49 to 14%; $P = 0.06$).

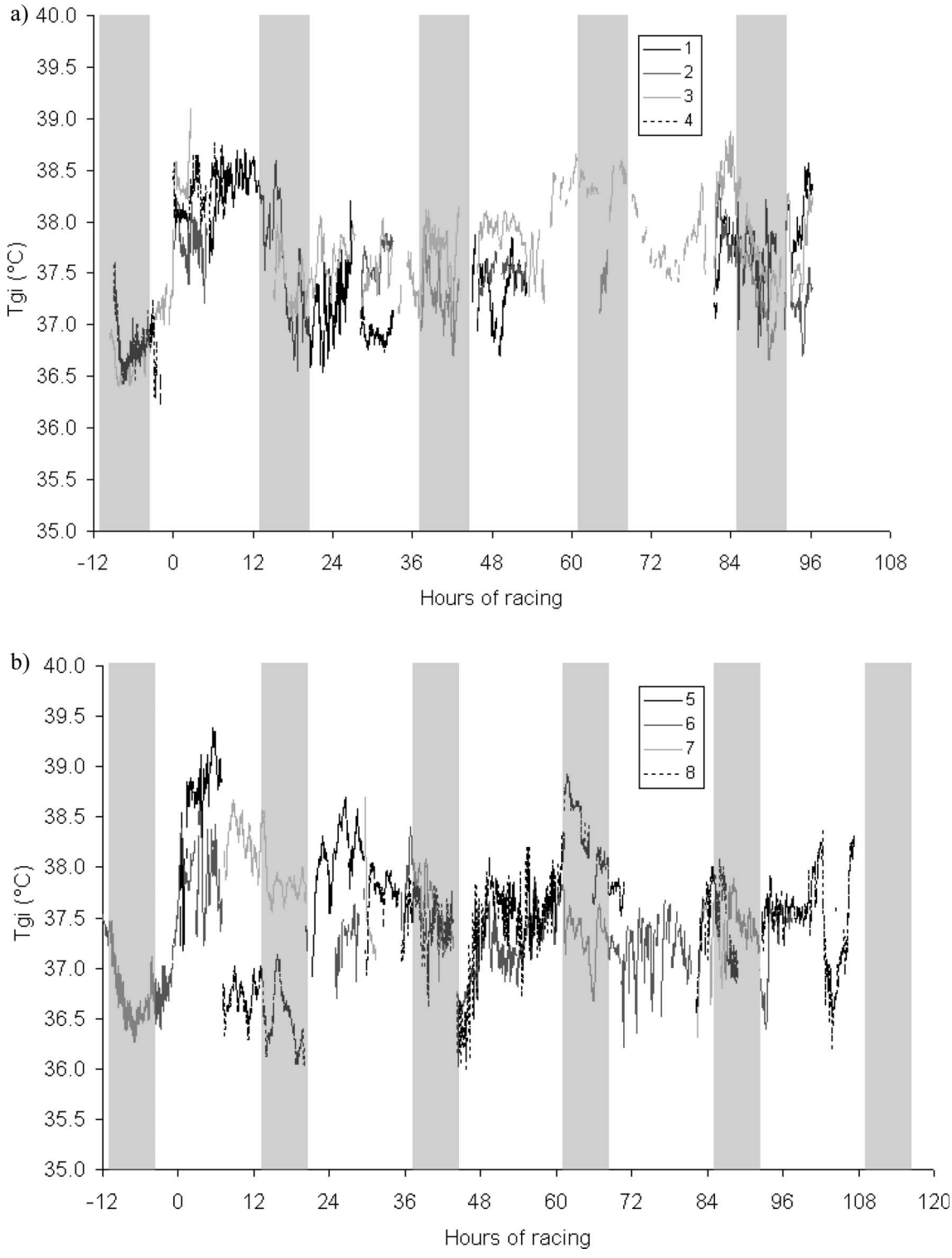


Figure 4. Gastrointestinal core temperature (Tgi) measured using a disposable thermometer pill for participants in teams Team A (a) and Team B (b). Shaded areas identify night periods (21:30–05:00 h).

## Discussion

This is the first comprehensive report of strain associated with very prolonged, competitive exercise and sleep deprivation characteristic of international adventure races. The five main findings were as follows: (1) within the first 24 h, exercise intensity was down-regulated to low to moderate levels (~40% $\dot{V}O_{2peak}$, inclusive of sleep and rest); (2) the heart rate–work rate relationship remained stable pre to post race, but perception of exertion with respect to work rate increased; (3) neither

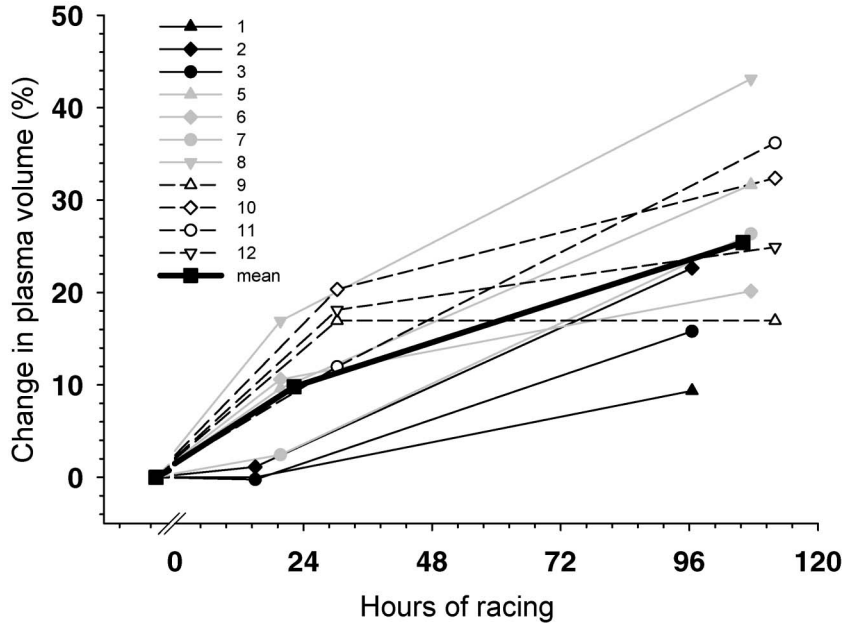


Figure 5. Plasma volume changes estimated from changes in haematocrit and haemoglobin concentrations in participants from three teams at 15, 20, and 30 h into the race, respectively, and at the finish.

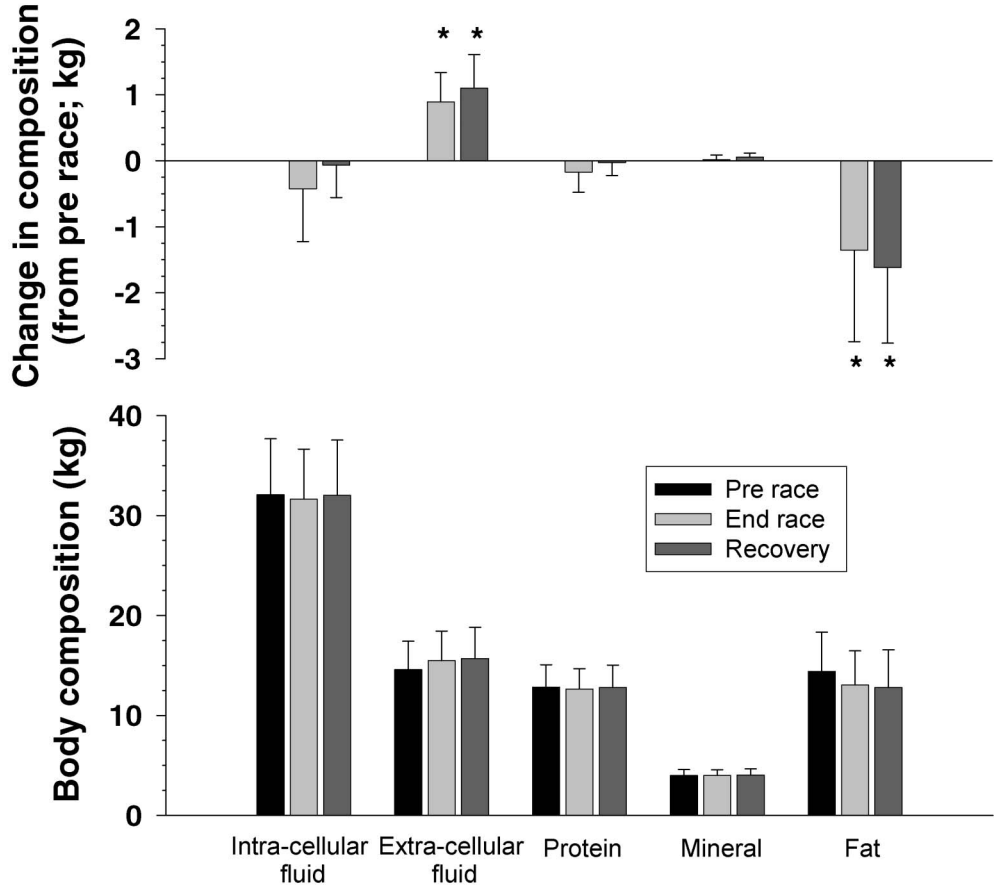


Figure 6. Multi-frequency bio-impedance analysis of participants ($n = 11$) rested and fasted 24 h before the race (pre race), within 60 min of finishing, and 24 h after the race (recovery). * $P < 0.05$ compared with pre race.

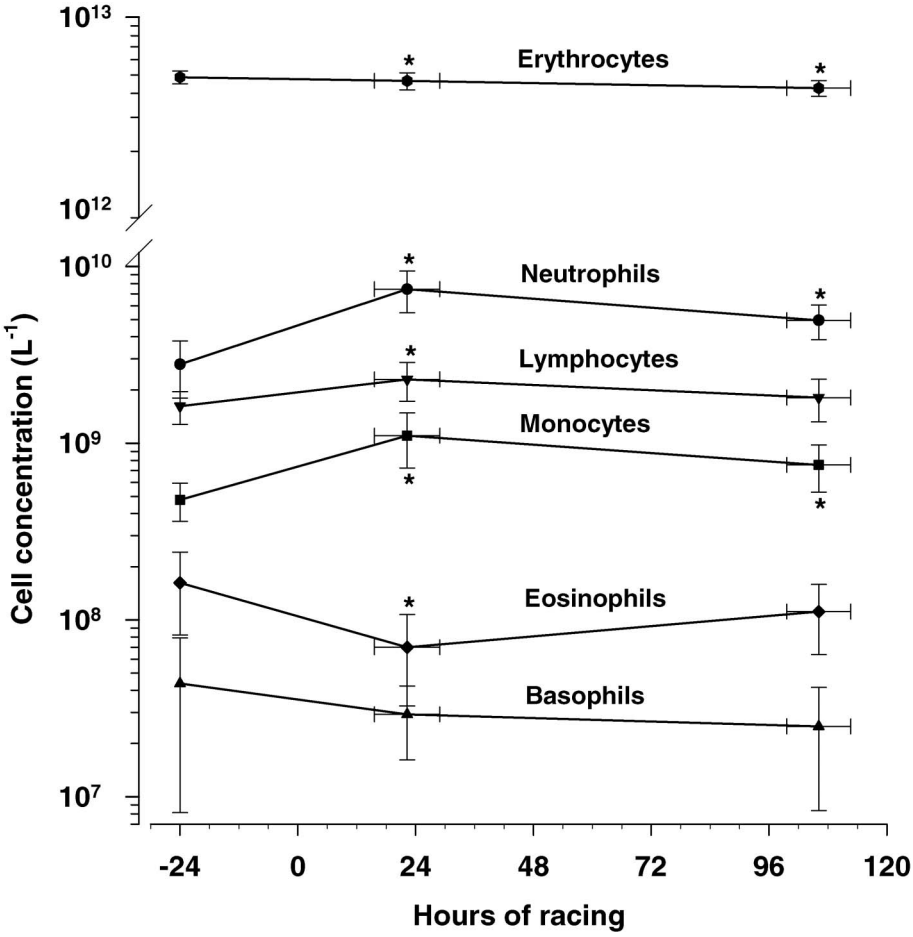


Figure 7. Pattern of change (mean $\pm$ *s*) of differential white blood cell counts in venous blood from athletes ($n = 11$) before, during (15 – 30 h), and after the race (96 – 116 h).* $P < 0.05$ compared with 24 h before the race.

clinically significant hypothermia nor hyperthermia was observed; (4) there was a 25% expansion in plasma volume; and (5) non-specific phagocyte-related cell numbers increased, yet unexpectedly lymphocyte numbers also increased.

Military-based studies provided the best comparison to the type of physical strain present in an adventure race. Soldiers can maintain 30 – 40% $\dot{V}O_{2max}$ work intensity for multi-day operations (Myles *et al.*, 1979; Myles & Romet, 1987; Opstad *et al.*, 1985; Opstad, Wiik, Haugen, & Skrede, 1994). However, military studies generally report more rest and/or sleep (i.e. 3 – 4 h per 24 h if longer than $\sim$60 h) than reported by our participants (1 – 2 h per 24 h). Despite the more severe sleep deprivation, our participants ''selected'' a similar work intensity for most of the race, following the initial 12 h of substantially higher intensity. The competitive nature of a race, the potentially greater intrinsic determination of adventure race competitors, and less load carried by racers compared with soldiers could contribute to this difference. Despite the cumulative sleep deprivation, mean work intensity was stable across days.

The observed exercise intensities presumably reflect a multi-factorial fatigue process involving feedforward and feedback mechanisms within the central nervous system (Bainbridge, 1919; Noakes, St. Clair Gibson, & Lambert, 2005). Self-selection of pace can be influenced by high or low body temperature (Gonzalez-Alonso *et al.*, 1999; Thompson & Hayward, 1996), pain (O'Connor & Cook, 1999; Surbey, Andrew, Cervenko, & Hamilton, 1984), liver, blood and/or muscle carbohydrate availability (Bergstrom, Hermansen, Hultman, & Saltin, 1967; Coyle *et al.*, 1983; McConell, Fabris, Proietto, & Hargreaves, 1994; Rauch, St. Clair Gibson, Lambert, & Noakes, 2005), depressed ventricular function (Ashley *et al.*, 2006), accumulation and/or depletion of metabolites and neurotransmitters in the central nervous system (Gandevia, 2001), and potential fatigue-mediating substances such as interleukin-6 (Robson-Ansley, de Milander, Collins, & Noakes, 2004), tryptophan, dopamine, and serotonin (Gandevia, 2001). A possible feedback or feedforward influence of factors such as perception of tiredness and mood state (e.g. ''vigour'' and ''fatigue'') (Scott, McNaughton, & Polman, 2006)

may be a consequence of the interactive effects of these multiple factors on behaviour.

The heart rate – work rate relationship was examined over a range of 0 – 50% peak power output (34 – 59% $\dot{V}O_{2peak}$) because it encompassed most of the heart rates obtained during racing, and was able to be completed by all participants post race. Despite the stable heart rate responses, the perception of effort increased, indicating that heart rate is not a reliable surrogate for perception of exertion, and vice versa. Perceived exertion increases with sleep deprivation (Martin, 1981; Soule & Goldman, 1973). Martin (1981) suggested that perceptual changes induced by sleep deprivation may be largely independent of the usual physiological cues correlated with heart rate and metabolic rate. Perceived exertion may have an important role in pace selection during an adventure race, but does not fully account for the slowing, as the extent of shift in the perceived exertion – work rate relationship is unable to account for the magnitude of fall in pace during racing. Perceived exertion may in some way reflect the biological demands associated with the maintenance of whole-body homeostasis (Noakes *et al.*, 2005). Perceived exertion during very prolonged, sustained, and sleep-deprived stress may be a more complete indicator of whole-body physiological and psychological strain than heart rate is, albeit a difficult one to validate against humoral fatigue mediators (Nybo & Secher, 2004).

Sleep deprivation and prolonged exertion also impair thermoregulatory processes. Sleep deprivation delays sweating (Dewasmes, Bothorel, Hoeft, & Candas, 1993) and attenuates its sensitivity (Sawka, Gonzalez, & Pandolf, 1984). Ultra-endurance exercise can impair cold defence, reducing vasoconstriction (Castellani *et al.*, 2001), tissue (fat and lean) insulation (Young *et al.*, 1998), and substrate availability (Gale, Bennett, Green, & MacDonald, 1981; Passias, Meneilly, & Mekjavic, 1996). In contrast, Castellani *et al.* (2003) found enhanced vasoconstriction and vigorous shivering activated at a lower threshold, such that heat debt was unaltered. Thus, the net effect of prolonged exercise and sleep deprivation on thermoregulation remains unclear. Body temperature in the present cohort was regulated such that no individual was recorded as clinically hypothermic (core $<35.0^{\circ}C$), and hyperthermia ($>39.0^{\circ}C$) was recorded only briefly, on day 1. This thermal stability might be anticipated given that humans thermoregulate using a proportional-control system relying mainly on powerful and elaborate behaviours (Hammel, 1968) in conjunction with vasomotor, thermogenic, and sudomotor mechanisms (Cabanac, 1975). Behavioural and autonomic responses remained adequate in this sleep-depriving and thermally challenging race. Heat-stressed competitors partially removed wetsuits during coasteering, whereas during a snow storm (third night) the same athletes continued trekking rather than sleep, reportedly to maintain heat production. Perhaps the exercise setting and situation influenced thermal tolerance and exercise adherence. Thompson and Hayward (1996) reported that 11 of 18 motivated, experimental participants could not tolerate 5 h walking in wet, windy, and cold conditions, despite core temperature averaging $36.9^{\circ}C$ at intolerance, a temperature well above clinically designated hypothermia and above some temperatures recorded during comparable conditions in our race setting. Thompson and Hayward (1996) highlighted the discomfort and psychomotor impediments of wetness and peripheral cooling, and suggested that the clinical definition of hypothermia may be inappropriate in an exercise setting, where body temperature is normally elevated. Recently, we have shown that endurance exercise may induce a fever-like elevation in body core temperature (Bradford *et al.*, 2007), a response that could exacerbate perceived coolness. Thus, we are unable to determine whether the adventure racers' measured thermal states were within ''reasonable'' limits, or simply reflected a situation in which they – unlike Thompson and Hayward's participants – were less able or willing to terminate the cold stress.

Plasma volume increased significantly, almost half of which occurred before 30 h when exercise intensity averaged more than 50% maximal power. There are limitations with our measurement technique, and postural shifts in blood volume would not have been complete by 3 – 5 min of sitting (which was the venous sampling time used here). However, this duration was similar before all sampling points (pre race, early race, post race), which would minimize systematic error, and any posture-related shifts in plasma volume could not account for the magnitude of change that we observed across the race (Harrison, 1985). Similarly, while participants' activity before sampling can influence the plasma volume, the within-race and end-race blood samples were obtained following very prolonged, upright exercise; situational factors that might lead to under- rather than over-estimation of the hypervolaemia. Finally, the bio-impedance, body mass, hormonal, and blood reticulocyte and electrolyte results collectively support the fluid expansion, and the expansion is remarkably similar to that observed following several days of exercise (4 – 10 h per day) in participants of widely varying endurance fitness (Fellmann *et al.*, 1999; Mischler *et al.*, 2003; Williams *et al.*, 1979). Thus, a marked hypervolaemic response seems reasonable.

In our results, expansion of the extracellular fluid did not reflect hyponatraemia and was mainly due to expansion of the plasma [four times greater

($P < 0.01$) than that of the wider extracellular fluid]. The mechanism(s) for plasma volume expansion could be orthostatically mediated increases in intravascular albumin (Mischler *et al.*, 2003) and electrolyte retention (Fellmann *et al.*, 1999; Williams *et al.*, 1979). Plasma volume expansion correlated moderately with increases in arginine vasopressin concentration. Arginine vasopressin is released as a response to exercise stress and therefore some water retention due to increasing arginine vasopressin concentration would be expected. However, arginine vasopressin concentrations, at race completion, were modest ($< 3$ pmol$\cdot$l$^{-1}$) and end-race urine specific gravities were not high, thus large perturbations of hydration status were not observed. We suggest that albumin synthesis, electrolyte retention, and the increase in arginine vasopressin may all have a role in mediating the plasma volume expansion. Finally, intracellular fluid volume appeared stable, in contrast to previous reports of an 8% expansion (Fellmann *et al.*, 1999) or 8% contraction (Williams *et al.*, 1979) following 7 days of endurance racing (6–10 h per day) and ''hill walking'', respectively.

Endurance exercise also alters immune status, partly via thermal and neuro-endocrine responses to stress (Bøyum *et al.*, 1996; Brenner, 2000; Shephard, Verde, Thomas, & Shek, 1991). Illness during adventure racing is common (Townes *et al.*, 2004). The present race was no exception; one member of Team A and four other teams (13%) withdrew due to acute illness, highlighting that pathogenesis can be a limiting factor in human performance (Bøyum *et al.*, 1996). The prolonged, sleep-deprived, and energy-deficit stress of military training has been shown to stimulate phagocyte-related (neutrophils and monocytes) cell numbers and functions, but suppress lymphocyte-related cell numbers and functions (Bøyum *et al.*, 1996; Brenner, 2000; Gomez-Merino *et al.*, 2003). Notwithstanding that our immune measures did not include spontaneous and stimulated leukocyte functions, we observed that the numbers of non-specific, phagocyte-related immune cells were elevated, but so were lymphocyte concentrations. The contrasting effect on lymphocytes seen here versus sustained exercise of ranger training (Bøyum *et al.*, 1996) may be attributed to higher cortisol concentrations recorded in ranger training, since adventure racing athletes do not incur the same energy deficit (Doel *et al.*, 2005). However, this would not explain the fall in eosinophil numbers. A more extensive investigation of immune responses to adventure racing is warranted.

A limitation of our study concerns the validity of expressing exercise intensity from heart rate. Errors occur in determining both maximum and minimum heart rates, and in heart rate responses to the different exercise modes and situational stressors. However: (i) errors in determining maximum and minimum heart rates would have opposing effects on the measured heart rate range; (ii) determining maximum heart rate using cycling provided an intermediate estimation between those of the other major exercise modes used in the race [running and kayaking (Kimber & Ross, 1996)]; and (iii) the energy expenditures of the athletes during this race, determined using doubly labelled water (Doel *et al.*, 2005), were higher than expenditures reported previously (Forbes-Ewan *et al.*, 1989; Hoyt *et al.*, 1991; Stroud *et al.*, 1993; Westerterp *et al.*, 1986), and support an essential inference from the heart rate measurements.

Adventure racing athletes settle into a race-sustainable pace approximating one-third to one-half of their aerobic capacity within the first 24 h of the race, irrespective of individual and team differences in fitness and experience. Important questions yet to be answered include why the athletes select a pace during the first 12 h that they cannot sustain, and what biological events cause the change in intensity to occur uniformly across the first day regardless of the remaining duration of the event.

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